synucleinopathy, e.g., Parkinson's disease. In one embodiment, the therapeutically effective amount is sufficient to prevent development of or alleviate to some extent one or more of the symptoms of the condition or disorder being treated, e.g., Parkinson's disease.

[0021] The term "subject" is defined herein to include animals, such as mammals, including but not limited to, primates (e.g., humans), cows, sheep, goats, horses, cats, rabbits, rats, mice, and the like. In preferred embodiments, the subject is a human. In some embodiments examples of any of the methods described herein, the subject is 40 years or older (e.g., 41 years old or older, 42 years old or older, 43 years old or older, 44 years old or older, 45 years old or older, 46 years old or older, 50 years old or older, 55 years old or older, 60 years old or older, 65 years old or older, 70 years old or older, 75 years old or older, 80 years old or older, 85 years old or older, 90 years old or older, or 41, 42, 43, 44, 45, 46, 47, 48, 49, 50, 51, 52, 53, 54, 55, 56, 57, 58, 59, 60, 61, 62, 63, 64, 65, 66, 67, 68, 69, 70, 71, 72, 73, 74, 75, 76, 77, 78, 79, 80, 81, 82, 83, 84, 85, 86, 87, 88, 89, 90, 91, 92, 93, 94, 95, 96, 97, 98, 99, 100, 101, 102, 103, 104 or 104 years old).

[0022] In some embodiments, the subject is a subject having a synucleinopathy, e.g., Parkinson's disease, suspected of having a synucleinopathy, e.g., Parkinson's disease or at increased risk of developing a synucleinopathy, e.g., Parkinson's disease (e.g., by virtue of family history, genetic testing, or presence of other identified risk factor).

[0023] In some embodiments, the subject does not present with a symptom (e.g., any of the symptoms described herein or known in the art) of a synucleinopathy neurological disorder (e.g., Parkinson's disease). In other embodiments, the subject has been diagnosed as having a synucleinopathy/neurological disorder (e.g., Parkinson's disease). In yet other embodiments, the subject has not been diagnosed as having a synucleinopathy disorder (e.g., Parkinson's disease).

[0024] In some embodiments, the subject has been diagnosed or identified as having a synucleinopathy neurological disorder (e.g., Parkinson's disease) that would benefit from treatment with a β2-adrenoreceptor agonist.

[0025] In some embodiments, the subject has previously been administered at least one dose of a therapeutic agent for a synucleinopathy, e.g., a Parkinson's therapeutic agent (e.g., any of the Parkinson's therapeutic agents described herein). In some embodiments, the subject is a participant in a clinical trial.

[0026] In other embodiments, the subject has been previously administered a different pharmaceutical composition and the different pharmaceutical composition was determined not to be therapeutically effective.

[0027] The term "population" when used before a noun means two or more the specific noun. For example, the phrase "a population of neuronal cells" means two or more neuronal cells.

[0028] The term "pharmaceutically acceptable salts" refers to salts of the active compounds that are prepared with relatively nontoxic acids or bases, depending on the particular substituents found on the compounds described herein.

[0029] As used herein, "obtain" or "obtaining" can be any means whereby one comes into possession of the sample by "direct" or "indirect" means. Directly obtaining a sample means performing a process (e.g., performing a physical method such as extraction or phlebotomy) to obtain a sample from the subject. Indirectly obtaining a sample refers to receiving the sample from another party or source (e.g., a third-party laboratory that directly acquired the sample). Thus, obtain is used to mean collection and/or removal of the sample from the subject. Some of the embodiments of any of the methods described herein can include obtaining a sample (e.g., a tissue biopsy) or samples from a subject.

[0030] Unless otherwise defined, all technical and scientific terms used herein have the same meaning as commonly understood by one of ordinary skill in the art to which this disclosure belongs. Methods and materials are described herein for use in the present disclosure; other, suitable methods and materials known in the art can also be used. The materials, methods, and examples are illustrative only and not intended to be limiting. All publications, patent applications, patents, sequences, database entries, and other references mentioned herein are incorporated by reference in their entirety. In case of conflict, the present specification, including definitions, will control.

[0031] Other features and advantages of the disclosure will be apparent from the following detailed description and figures, and from the claims.

SEQUENCE LISTING

[0032] The instant application contains a Sequence Listing which has been submitted electronically in ASCII format and is hereby incorporated by reference in its entirety. Said ASCII copy, created on Apr. 20, 2018, is named "Sequence Listing" and is 2,353 bytes in size.

DESCRIPTION OF DRAWINGS

[0033] FIG. **1**A is a schematic representation of a work flow leading to the identification beta-adrenoreceptor 2 (β2AR) as regulator of SNCA neuronal gene expression (top panel). "Campaign view" of compounds screened in the cell-based endogenous SNCA mRNA abundance (y-axis) observed in the drug-treated human neuroblastoma cells compared to DMSO-treated human neuroblastoma cells; housekeeping gene UBC was used to control for input RNA)

[0034] FIG. **1**B shows the chemical structure, FDA approval, indication and blood-brain penetrance of three selective β2AR compounds.

[0035] FIG. **1**C is a bar graph showing the relative endogenous Snca mRNA abundance in rat primary cortical neurons (n=4) after exposure to PAR agonists metaproterenol (5 μM) clenbuterol (20 μM) and salbutamol (10 μM).

[0036] FIG. **1**D is a bar graph showing the relative endogenous α-Syn protein abundance in rat primary cortical neurons (n=4) after exposure to β2AR agonists metaproterenol (5 μM), clenbuterol (20 μM) and salbutamol (10 μM).

[0037] FIG. **1**E is a bar graph showing the relative endogenous Snca mRNA abundance in a dose-dependent manner in neuroblastoma cells (N=6-8) after four days of clenbuterol treatment (5, 10, 20 μM). *P<0.05,**P<0.005, one-way ANOVA with Tukey's; means±standard error (SEM) are shown.

[0038] FIG. **1**F is a bar graph showing the relative endogenous α-Syn protein abundance in a dose-dependent manner in neuroblastoma cells (N=6-8) after four days of clenbuterol treatment (5, 10, 20 μM). *P<0.05, **P<0.005, one-way ANOVA with Tukey's; means±SEM are shown.

[0039] FIG. **1**G is a bar graph showing relative SNCA mRNA abundance in human SK-N-MC cells treated with